An electrophysiological *Monetary Incentive Delay* (e-MID) task: A way to decompose the different components of neural response to positive and negative monetary reinforcement

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Abstract

Background: The ability to anticipate and then secure future rewards and avoid future punishments by responding effectively to environmental demands is at the core of successful decision making. Disruptions to these processes have been shown to be implicated in a number of psychiatric conditions. In the current paper we use the electrophysiological monetary incentive delay task (e-MID) to decompose the neural response to (i) reinforcement anticipation, (ii) reinforcement-contingent target processing and (iii) reinforcement-related feedback.

Methods: Thirty-eight adolescents and young adults performed an ERP-based analogue of the monetary incentive delay task. ERP components previously associated with motivationally salient cue (cue-P3 and contingent negative variation, CNV), target (P3) and feedback (success vs. failure; feedback-related negativity; FRN and the late positive potential; LPP) stimuli were examined.

Results: Response times were shorter and less variable in the *monetary gain* and *loss* conditions. Distinctive ERP components were observed for each phase of reinforcement processing. First, cue-P3 was enhanced to *monetary gain* cues. Predicted alterations in cue-P3 following *monetary loss* cues and the CNV following cues of either *monetary loss* or *gain* were not observed. Target P3 was enhanced in both incentive conditions. The FRN was greater following *monetary loss* feedback. LPP amplitude was enhanced following feedback denoting *monetary gain* and the avoidance of *monetary loss*.

Conclusion: Although behaviourally the effects of monetary loss and gain were similar, the e-MID task differentiated neural processing in terms of anticipation and feedback-related brain potentials. The e-MID task and the results of the current study provide a valuable complement to fMRI-based approaches to studying normal and abnormal brain correlates of reinforcement processing.

Key words: reward, punishment, event-related potential, reinforcement, anticipation, outcome

Research Highlights

- Cues of monetary gain and loss reinforced shorter and less variable response times in an ERP analogue of the Monetary Incentive Delay (MID) task
- Reinforcement anticipation was characterised by an enhanced cue-P3 to gain cues, whilst CNV amplitude was not altered by monetary reinforcement
- Target P3 was enhanced in both monetary gain and loss conditions. Feedback relating to monetary loss elicited an enhanced FRN
- The LPP was most sensitive to feedback denoting monetary gain and the avoidance of its loss
- The results from the e-MID task complement current fMRI-based research investigations of typical and atypical reinforcement processing.

1. Introduction

The ability to accurately anticipate reinforcement, secure it through effective performance and be sensitive to its delivery is a fundamental aspect of adaptive behaviour and learning (for a comprehensive review see Luhmann, 2009). Increasingly, the disruption of these normal reinforcement mechanisms is considered to be core to a number of serious forms of mental disorder. These include attention-deficit hyperactivity disorder (ADHD, Plichta, et al., 2009; Scheres, et al., 2007; Ströhle, et al., 2008; Van Meel, et al., 2011), substance abuse (Beck, et al., 2009), schizophrenia (Juckel, et al., 2006) and depression (Forbes, et al., 2012). The potential importance of altered reinforcement mechanisms in mental disorder has prompted recent scientific focus and investigation of the neural correlates of normal and abnormal reinforcement processing in an attempt to isolate the underlying causes of disorder and to identify new and more effective treatments (Sonuga-Barke & Halperin, 2010). Positron Emission Tomography (PET) and functional magnetic resonance imaging (fMRI) work have provided important insight into the underlying neurobiology of reward processing, and in particular have implicated a network of dopamine-modulated brain circuits including the nucleus accumbens (NAcc), caudate, putamen, thalamus, medial orbitofrontal cortex (OFC), bilateral anterior insula and the posterior cingulate cortex (Knutson & Cooper, 2005; Knutson, et al., 2001; Knutson, et al., 2000; Liu, et al., 2011; McClure, et al., 2007; McClure, et al., 2004).

A variety of experimental paradigms have been employed to understand reinforcement processing across different forms of mental disorder and in healthy individuals. These paradigms are distinct in three important respects. Firstly, the tasks differ in whether a cue to signal the possibility of an upcoming reward is present or absent. Secondly, they differ in whether the participant is led to believe that reinforcement is contingent on their performance (i.e., the speed of their response) as opposed to probability and chance (i.e., guessing tasks). Thirdly, while many studies only consider the effect of gaining a reward (versus the failure to gain a reward), other studies also consider the effect of loss (versus the avoidance of loss). Guessing tasks are frequently used in this field of research and typically include a comparison of reward and no reward or loss conditions in the absence of cues. Examples of these tasks include the 'virtual maze task' (see Baker & Holroyd, 2009; Holroyd et al., 2008) and the 'balloon gain context task' (see Crowley et al., 2008; Holroyd, et al., 2003; Larson et al., 2011). In the virtual maze task, participants are presented with a virtual T-maze consisting of a stem that intersects two alleys at right angles; participants are instructed to select the alley (right or left) that they believe contains a reward and, following their selection, they are provided with one of two randomly selected visual feedback stimuli to indicate whether they have gained a

monetary reward or failed to gain a reward (i.e., the probability of gaining a reward is 50%). Similarly, the balloon gain context task requires participants to choose one of four coloured balloons on every trial; they are informed that one of the balloons contains a monetary reward, whereas the other three are associated with a monetary loss. In line with the virtual maze task, visual feedback signalling gain or loss are presented randomly on every trial such that there is a 50% chance of winning or losing money. In contrast to guessing tasks, alternative paradigms have been developed in which a cue indicates the possibility of an upcoming reward and participants are informed that they can maximise rewards and minimise losses by responding as quickly and accurately as possible (e.g., Cohen et al., 2012; Holmes & Pizzagalli, 2010; Knutson et al., 2001); these tasks are important in considering an individual's motivation to gain rewards or avoid losses by improving their performance. One of the most influential experimental paradigms of this type has been the Monetary Incentive Delay (MID) task which was developed by Hommer and Knutson (Knutson et al., 2001; Knutson et al., 2000).

The MID task was designed to be used in conjunction with fMRI, to differentiate between the neural response to (i) the anticipation of reinforcement and (ii) its delivery following successful performance (Knutson et al., 2001; Knutson et al., 2000). In the MID task, participants are presented with a cue which signals the opportunity to win or lose money. This cue is followed, after a variable delay, by a target prompting a sufficiently rapid response to win or avoid losing money. Feedback relating to the success or failure of this response and the associated monetary outcome is then presented. Perhaps one of the most scientifically appealing features of the MID task is its elegant simplicity: an informative cue precedes a perceptually undemanding target detection task requiring only a button press response, and this sequence of events is then followed by an uncomplicated system of feedback. As a result, the MID task has been used in a large number of fMRI studies to successfully delineate between core networks of reinforcement-related brain regions recruited during different stages of processing (i.e. anticipation and outcome, for a meta-analysis see Liu et al. 2011). Contradicting initial reports using the MID task which have linked the activation of NAcc specifically with reward anticipation (Knutson et al., 2001; Knutson et al., 2000), the results of a recent meta-analysis instead suggested that activation of this region may be observed during both anticipatory and outcome stages of reward processing (Liu et al., 2011). The OFC, in contrast, was most reliably associated with reinforcement evaluation and outcome, while both the NAcc and OFC were typically enhanced for positive compared to negative outcomes (Liu et al., 2011). In contrast, negative

reinforcement has been associated with enhanced activation of the anterior insula (Liu et al., 2011) and thalamus (Knutson et al., 2000).

The utility of the MID task with regard to the differentiation of neural substrates linked with reinforcement anticipation from outcome has been valuably demonstrated with regards to isolating abnormalities in reinforcement processing associated with psychopathology. For example an attenuated neural response during reward anticipation has been reported in patients with ADHD (Plichta et al., 2009; Scheres et al., 2007; Ströhle et al., 2008), detoxified alcoholics (Beck et al., 2009), obsessive compulsive disorder (Figee, et al., 2011), schizophrenia (Juckel et al., 2006) and major depressive disorder (Forbes et al., 2012) as well as children who have been maltreated (Dillon, et al., 2009; Mehta et al., 2009). While in contrast, chronic cannabis users are found to exhibit exaggerated right ventral striatum activation during the anticipation of incentives (Nestor, et al., 2010). On the other hand, following informative feedback relevant to reinforcement outcome, activation of reward-related brain circuitry to monetary gain is found to be increased in patients with ADHD (Ströhle et al., 2008) and substance dependent patients (Bjork, et al., 2008); while activation of reward related brain areas in patients with schizophrenia (Waltz, et al., 2010), patients with autism spectrum disorder (Dichter, et al., 2012) and patients with major depressive disorder (Pizzagalli, et al., 2009) is found to be decreased. The differences between and within mental disorders in the activation of reward-related brain regions during anticipatory and reward receipt stages underscore the importance of using tasks like the fMRI MID to dissociate the neural response associated with each stage of reinforcement processing.

Despite its obvious advantages in terms of temporal resolution and the potential for fine grained differentiation of the neural response to reinforcement, less work has employed event-related electrophysiological approaches to understanding normal and abnormal reinforcement processing (although see Brunia, et al., 2011; De Pascalis, et al., 2010; Goldstein, et al., 2006; Goldstein, et al., 2008; Ohgami, et al., 2006; Van Meel et al., 2011). Specifically, electrophysiological methods offer a more exact neuroimaging platform with which to decompose the neural response to anticipatory and outcome stages of reinforcement processing precisely. Electrophysiological methods are also more child- and patient-friendly than most other brain imaging methodologies. In the current study we exploit the superior temporal resolution and direct association between underlying neural activity and scalp recorded EEG, and use a variant of the MID task (Electrophysiological or e-MID) to investigate event-related brain potentials (ERP) to reinforcement cues, reinforcement-contingent target processing and reinforcement-related feedback with

both positive (securing gain) and negative (avoiding loss) monetary reinforcement. Previous work on the ERP correlates of reinforcement processing (Brunia et al., 2011; Carlson, et al., 2011; De Pascalis et al., 2010; Goldstein et al., 2006; Goldstein et al., 2008; Groom, et al., 2010; Holroyd & Coles, 2002; Van Meel et al., 2011) have allowed us to identify a number of candidate brain potentials of interest. To the best of the authors' knowledge however, the current study represents the first attempt to decompose the event-related neural response to anticipatory and outcome phases of reinforcement processing using the e-MID task.

First, with regards to the anticipatory neural response to processing of *cues* prior to task performance, previous work has highlighted the sensitivity of the P3 component to reinforcement (Goldstein et al., 2006; Goldstein et al., 2008). The cue-P3 is a centroparietal positivity which emerges between 300-600 milliseconds post-stimulus, and increases as a function of reinforcer magnitude (Goldstein et al., 2006). A second component, the contingent negative variation (CNV) has received less attention with regards to the anticipation of reinforcement, despite its role in anticipatory attention and preparation related to effortful processes (Falkenstein, et al., 2003; Gómez, et al., 2007). The CNV is a slow negative potential, maximal over frontocentral sites and elicited by an informative cue signalling the impending presentation of a stimulus requiring a response (Walter, et al., 1964). The CNV consists of two sub-components, an early wave ('orienting' O wave) related to the alerting properties of the cue, and a later component ('expectancy' E wave) which is associated with anticipation of the target stimulus and the engagement of effortful processes associated with the required response (Brunia et al., 2011; Brunia & Vingerhoets, 1981; Van Boxtel & Bocker, 2004). There is evidence to suggest the CNV is modulated by motivation (Cant & Bickford, 1967; Irwin, et al., 1966), effort (Falkenstein et al., 2003; Gómez et al., 2007) and the anticipation of affective or motivationally salient stimuli (Baas, et al., 2002; Klorman & Ryan, 1980). Despite its motivational salience, other work has reported no effect of varying monetary reward on this component (Goldstein et al., 2006).

Second, related to *target-related* brain potentials, there is a substantive body of evidence to suggest that the centroparietal P3 component elicited approximately 300 ms following a target stimulus is a robust index of task-relevant and motivated attention (for a review see Polich & Kok, 1995). In a recent study examining the effect of monetary reward on performance in children with and without ADHD, Groom et al. (2010) reported enhanced target-P3 amplitude in a reward and punishment condition relative to a control

condition, although this component was not found to differ between positive and negative reinforcement types.

Finally, in terms of the neural response to informative *feedback* about rewarding or punishing outcomes, two ERP components in particular have been shown to be sensitive to motivationally salient outcomes. First, the frontocentral feedback-related negativity (FRN) is enhanced by negative performance feedback (Miltner, et al., 1997), and thought to reflect an alerting signal following unexpected and unfavourable feedback (e.g., monetary loss or omission of monetary gain, Holroyd & Coles, 2002; Luu, et al., 2000). Second, the P3 component elicited by informative feedback is considered a robust marker of reinforcement magnitude, although it is thought to be less sensitive to reinforcement valence (i.e. reward or punishment see Yeung & Sanfey, 2004). The P3-like component elicited by motivationally and emotionally salient outcomes such as reward, is often referred to as a Late Positive Potential (LPP) and argued to reflect the affective processing of visual stimuli. Consistent with this notion, the LPP is generated in occipital and parietal cortices (Keil, et al., 2002; Sabatinelli, et al., 2007), both of which receive projections from the amygdala, a brain structure known to be involved in emotional processing (Bradley, et al., 2003). The LPP is reliably enhanced by positive and negative valanced stimuli, and is positively associated with the intensity of valenced visual stimuli (for a review see Hajcak, et al., 2010).

Importantly, few ERP studies have compared the effects of positive and negative reinforcement directly, especially in relation to the anticipation of reinforcement. One study which has compared positive and negative reinforcement cues, reported an enhanced P3 response to cues signalling potential monetary loss relative to potential reward (Löw, et al., 2008). With regards to target processing, a differential P3 response during positive or negative reinforcement has not been reliably observed (e.g., see Groom et al., 2010). Most research however has focused on the neural response to positive or negative reinforcing feedback (Boksem, et al., 2008; De Pascalis et al., 2010; Van Meel et al., 2011). This work has served to highlight the sensitivity of the FRN to negative compared with positive outcomes (Holroyd & Coles, 2002; Holroyd, et al., 2006; Wu & Zhou, 2009). In contrast, the evidence pertaining to the sensitivity of the feedback-related P3 or LPP component to reinforcement valence is mixed. Some researchers have reported an enhanced response following positive outcomes (Wu & Zhou, 2009; Yeung, et al., 2005), while others have reported a larger response following negative outcomes (De Pascalis et al., 2010), and others still report no difference (Yeung & Sanfey, 2004). In the current study we compared the neural response associated with positive and negative reinforcement during anticipatory, target and outcome stages.

The primary goal of this study was to develop an electrophysiological analog of the MID task (the e-MID) and to use this task to decompose the event-related neural response to positive and negative reinforcement during anticipatory, target and outcomes stages of reinforcement processing in a population sample with a broad age range. Our predictions were as follows. In accord with fMRI-based MID research, we predicted that cues signalling the opportunity to win or avoid losing money would reinforce faster and less variable response times (RT) and at the neural level we predicted that these cues would elicit enhanced anticipatory brain activity (i.e., cue-P3 and CNV) as participants prepared to respond faster. In relation to feedback about reinforcement outcome we predicted (i) that the neural response to the unfavorable outcome of monetary loss would result in an enhanced FRN when compared with negative feedback in a feedback-only control condition, and (ii) the affective salience of winning or actually losing money would elicit an enhanced LPP. Importantly, this study also set out to directly compare the electrophysiological response to positive and negative reinforcement types at each stage of processing. Here we predicted larger cue P3 amplitude to negative compared with positive reinforcement cues and a larger FRN to negative reinforcement outcomes. Positive and negative reinforcement conditions were not predicted to elicit differences in the target P3; and in view of the mixed findings concerning the LPP, clear predictions relating to the sensitivity of this component to the valence of reinforcement outcomes were not readily apparent. Finally, we examined the neural response to reinforcement in two groups of individuals differing in age and gender to explore the robustness of the effects across these factors.

2. Methods and Materials

2.1 Participants

Eighteen young adults who were undergraduate students from the School of Psychology, (2 male, mean age: 21.9 years, SD: 4.5 years); and 20 adolescents from local schools within Hampshire (all male, age: 15.0 years, SD: 0.7 years) took part in the study. Participants had not taken any medication for any condition for at least 24 hours prior to the experiment, and all participants had normal or corrected vision and hearing. The research protocol was approved by the University of Southampton, School of Psychology Ethics Committee and the Southampton and South west Hampshire Research Ethics Committee B. All adolescent participants completed two subscales of the *Wechsler Intelligence Scales for Children* (WISC-III; Wechsler, 1991) and all IQ scores feel within the normal range (M: 99.3, SD: 15.2).

2.2 Experimental Paradigm and Procedure

All participants were familiarised with the electrophysiology laboratory and EEG recording procedure before informed consent was taken. Consent was taken from both young adult and adolescent participants, although parents/guardians of all adolescent participants were also asked to give consent for their child's participation in the current study. Participants completed a short screening questionnaire to assess vision problems, medication and psychotropic substance use, and neurological disorders. They were then fitted with recording electrodes and seated in a comfortable chair in the testing room approximately 80 cm away from the monitor in a darkened room. Participants took part in two core assessments: an e-MID task (Knutson et al., 2000) and an escape from delay incentive (EDI) task (Broyd et al. 2012, Lemiere et al. 2012). Undergraduate participants also completed an approach/avoidance task. The approach-avoidance and EDI task were completed as part of another experiment and will not be reported here. The order of the tasks was counter-balanced. Participants were instructed to sit as still as possible and to minimise blinking.

At the start of each trial, participants were presented with one of three blue cue stimuli which was followed shortly afterwards by the target stimulus, a white square (see Figure 1). Participants were instructed to respond to the target as quickly as possible with the thumb on their dominant hand via a button box key. Feedback was provided - a green tick signalled that their response was 'fast enough' while a red cross signalled that their response was 'too slow'. This task included an adaptive algorithm which tracked each participant's response on a trial by trial basis and adjusted the response window for a 'fast enough' response so that all participants received positive feedback, based on their own performance, on 66% of trials. This also ensured that all participants won the same amount money (each participant received £10). There were three trial types in each session which were presented with equal probability and in random order: *Gain, Loss* and *Feedback only* (a neutral control condition). In the *feedback only* condition (signalled by the blue cross cue) participants were told that although they would receive feedback about the speed of their response, they could neither win nor lose money on these trials. In the *Gain* condition (signalled by a blue octagon cue) participants were informed that a fast response would receive positive feedback and participants would gain 5 points (i.e., 20 pence) on each of these trials, while a slow response would receive negative feedback and no points or money would be gained. In the *Loss* condition, (signalled by a blue diamond cue), participants were informed that a fast response would receive positive feedback and avoid the loss of 5 points (i.e., 20 pence), while a slow response would receive negative feedback and incur a loss of 5 points (i.e., 20 pence) on each of these trials. Participants were told that they

would receive their total winnings in cash at the end of the last block of the task. A practice block of 30 trials was completed prior to the experimental blocks to allow participants to learn the association between each cue and experimental condition. The cue and target stimuli were each presented for 250 ms for the young adult sample, and separated by a fixed 2000 ms inter-stimulus interval (ISI). For the adolescent sample, the ISI was varied randomly between 2000 and 2500 ms (M: 2250 ms). The feedback stimuli were presented for 1500 ms and appeared on screen 1450 ms following the offset of the target stimulus. Young adult participants completed 3 experimental blocks of 100 trials, and were given a break between each block. Adolescent participants completed 3 experimental blocks of 60 trials, and were given a break between each block.

2.3 Electrophysiological Acquisition and processing

An electrode cap (Easycap, Herrsching, Germany) containing 66 equidistantly spaced silver/silver chloride (Ag/AgCl) electrodes was fitted to each participant and EEG data was recorded using Neuroscan Synamps^2 70 channel EEG system, DC-coupled recording equipment. The data were sampled at 500 Hz with a low pass filter at 70 Hz and referenced to an electrode on the nose. A ground electrode was fitted midway between the electrode at the vertex and frontal sites. Vertical electro-oculogram (vEOG) was recorded from four electrodes: two bipolar electrodes were placed directly beneath the left and right eyes and affixed with tape, while the two electrodes placed above the right and left eye were included within the electrode cap. Impedances for vEOG, reference and cap electrodes were kept below 5 kΩ. The continuous EEG data were transformed using a DC offset and linear detrend algorithm in neuroscan (Scan 4.4). On each trial ERP data were examined to the cue, target and feedback (positive vs. negative) stimuli. The ERP epoch to the cue began 200 ms prior to stimulus onset and ended 2000 ms post-stimulus presentation. The ERP epoch for the target included 200 ms pre-stimulus activity and 800 ms post-stimulus activity, whilst the epoch for the feedback stimulus also included 200 ms pre-stimulus activity and extended 1500 ms post-stimulus. An ocular artifact reduction procedure (Semlitsch, et al., 1986) based on left eye vEOG activity was used to remove blink artifacts and other eye-movements from the ERP data. Further, any epoch that exceeded ± 100 μV at any non-frontal scalp site was rejected. ERP data to all stimuli were baselined to the 200 ms pre-stimulus interval and filtered 48 dB down at 32 Hz using a low-pass filter for analysis. Individual ERP averages were based on a minimum of 20 trials with the exception of negative feedback trials in which a criterion of a minimum of 10 trials was used (cue M: 55 trials, SD: 20 trials; target: M: 60 trials, SD: 14 trials; positive feedback: M: 40 trials, SD: 10 trials; negative feedback: M: 19 trials, SD: 5 trials). Consistent

with these criteria, data from two young adults and two adolescents were excluded from all analyses. A further two adolescents were excluded for ERP analyses to the cue, one young adult was additionally excluded from the analyses pertaining to the target, and a further two young adults were excluded for analyses relating to negative feedback. Because a minimum of 10 trials could be considered insufficient to produce reliable ERPs, an analysis of signal to noise ratio (SNR) was performed using the computational process available in Neuroscan 4.4. Specifically, SNR is calculated as the ratio between the variance of the 'noise' in the pre-stimulus interval and the variance of the 'signal' in the post-stimulus interval (i.e. signal/noise). Using this method, SNR was calculated for every participant at electrode sites 1, 5, 13 and 25 for Cue P3, Target P3 and LPP; sites. 1, 2, 3, 7, 8 and 18 for the CNV; and SNR was calculated at sites 1 and 2 for the FRN. The SNR for each component was then averaged across electrode sites for each component (see Table 1 for SNR mean and standard deviation). To examine possible differences in SNR between conditions, repeated measures ANOVA with condition (feedback only control, monetary gain, monetary loss) as the within-subjects factor and group (young adults, adolescents) as the between-subjects factor were performed for Cue P3, CNV, Target P3 and FRN SNR data. SNR was not found to differ between conditions for Cue P3, CNV Target P3 or the FRN (all p values > 0.100), and group was not found to interact with condition for an ERP component (all p values > 0.100). For the LPP, a repeated measures ANOVA was performed with condition (feedback only control, monetary gain, monetary loss) and feedback type (positive, negative) included as within-subjects factors and group (young adults, adolescents) as the between-subjects factor. This analysis did not reveal an effect of feedback type on SNR, nor was group found to interact with condition or feedback type (all p values > 0.100). A main effect of condition was observed ($F(2,66) = 6.72$, $p = .002$) and post-hoc analyses revealed significant lower SNR in the feedback only control condition relative to the Monetary Gain ($p = .005$) and Loss ($p = .002$) condition. However, because SNR values were on average greater than 2 for all ERP components and across conditions, these data were considered acceptable for further analysis (Luck, 2005).

Following the cue stimulus, a clear cue P3 component could be observed from around 350 ms and the CNV emerged around 600 ms. Cue P3 was quantified as the mean amplitude between 350 and 600 ms at midline central and parietal sites (electrode sites 1, 5, 13 and 25), while the CNV was quantified in two time windows (young adult: early CNV: mean amplitude between 600 and 1100 ms and late CNV: mean amplitude between 1100 and 1600 ms; Adolescent: early CNV1: 650 and 1150 ms and late CNV2: 1150 and 1650 ms) at six frontocentral sites (electrode site 1, 2, 3, 7, 8 and 18). For all electrode site locations

please refer to Figure 1. The target-P3 component was also identified and quantified as the mean amplitude between 250 and 450 ms in the young adult sample and 275 to 450 ms in the adolescent sample at midline central and parietal sites (electrode sites 1, 5, 13 and 25). Following the onset of the feedback stimulus a FRN emerged at midline frontocentral sites at around 230 ms, and this component was quantified at two sites (electrodes 1 and 2) as the mean amplitude between 230 and 300 ms for the young adults and 230 to 290 ms in the adolescent sample. The LPP emerged around 300 ms at midline central and parietal sites (electrode sites 1, 5, 13 and 25), and was analysed in two time segments and early LPP (LPP1, mean amplitude between 320 and 450 ms in young adults, 290 and 450 ms in the adolescent sample) and a late LPP (LPP2, mean amplitude between 450 and 600 ms in both samples).

2.4 Data analysis

Preliminary analyses were conducted to examine the impact of a range of procedural and demographic variables that did not constitute the main focus of the study: gain/loss vs. feedback-only control condition and group (adolescent, young adults), component time window (CNV 1 and CNV 2 or LPP1 and LPP2) or electrode site. Repeated measures ANOVAs were with group as a between-subjects factor and condition (gain/loss vs. feedback-only control condition), electrode site, and time window were entered as within-subjects factors. Only where these factors consistently interacted with condition were they retained for main analysis. RT data were trimmed to remove responses which were faster than 150 milliseconds and exceeded +/- 2.5 SD around the mean response time. ERP data were also examined for outliers (+/- 2.5 SD around the mean) and all analyses were repeated with and without identified outliers. For all cases the pattern of effects remained the same and therefore ERP data were retained in original form. ERP data were also checked for sphericity and Greenhouse-Geisser corrected values were reported for any violation.

In the main analysis, ANOVAs with condition as the within-subject variable were performed to examine MRT, SD of RT, Cue P3, CNV and target P3. An identical analysis was performed for FRN data but was conducted for the negative feedback condition only. For the LPP this analysis was carried out for both positive and negative feedback separately. Finally, where significant effects of *both* gain and loss were observed for ERP or performance data, these were compared directly with each other after controlling for the feedback only condition using a repeated measures ANOVA with incentive condition (gain vs. loss) as the within subject factor and entered the feedback-only condition as a covariate.

3. Results

3.1 Monetary Gain:

There was no interactions between condition and group for MRT, Cue P3, CNV, Target P3, LPP or FRN components. An interaction between condition and group was identified for SD of RT ($F(1, 36) = 6.06$, $p = .019$), however post-hoc tests revealed a significant effect of condition for both groups. Condition did not interact with CNV time-window, although a significant interaction was observed for LPP following positive feedback ($F(1, 32) = 11.53$, $p < .001$). Post-hoc tests indicated that the effect of condition was significant in both LPP time windows. Finally, electrode site was not found to interact with condition (or group) for any ERP measure, except the LPP following positive feedback ($F(1.73, 96) = 4.87$, $p = .012$), here too, post-hoc tests demonstrated a significant effect of condition at every electrode. Given these findings, group, electrode site and time window were dropped from the analysis. ERP waveforms at each site and for each group separately to the cue, target and feedback are shown in Appendix A to F.

Table 2 reports all statistical comparisons for the main analysis, as well as mean (standard deviation) performance and ERP data for the gain and loss condition relative to the feedback only control condition. The opportunity to win money was found to have a significant effect on task performance, such that RTs were significantly shorter and less variable in the gain compared to the feedback only condition. Cue- and target-P3 were also enhanced in the monetary gain compared to feedback only control condition (see Figure 2 and Table 2), while no effect of condition was observed for the CNV. Finally, FRN and LPP amplitude did not differ for feedback signifying omitted gain (negative feedback signifying no money had been won) and negative performance-only feedback. Instead, LPP amplitude was enhanced following feedback signifying actual monetary gain relative to positive performance-only feedback.

3.2 Monetary Loss:

There were no significant interactions between condition and group for any task performance or ERP variable. Condition did not interact with electrode site for any ERP component, and no interaction was observed between condition and time-window with the exception of the LPP following positive-feedback ($F(1, 32) = 4.86$, $p = .035$). A significant effect of condition for both time-windows was confirmed via post-hoc tests, and given the pattern of results these factors were dropped from the analysis.

The main analysis revealed faster and less variable response times when there was an opportunity to lose money relative to the feedback only control condition (see Table 2 for statistical comparisons,

condition means and standard deviations). An effect of condition on anticipatory neural responses (cue P3 and CNV) was not observed, although target P3 was significantly enhanced when there was an opportunity to lose money (see Figure 2). Negative feedback signalling actual monetary loss elicited larger FRN and tended to elicit larger LPP amplitude when compared with negative performance only feedback, while the successful avoidance of monetary loss (positive feedback) also elicited a larger LPP response (Figure 3).

3.3 Comparison between effects of Gain and Loss: The final analysis compared the effects of monetary gain and loss directly. An effect of both the gain and loss conditions was identified for four measures – performance (MRT and SD of RT), target P3 and LPP following positive feedback. Direct comparison of these effects (when the feedback only condition was entered as a covariate) did not reveal any significant differences between the effects of gain and loss (all p values > 0.100).

4. Discussion

To our knowledge this is the first study to demonstrate the utility of an e-MID task in the decomposition of the electrophysiological brain response to positive and negative reinforcement during anticipatory, target and outcome stages of reinforcement processing. We made a number of predictions. Consistent with our first prediction and in accord with findings from fMRI-based MID research, cues signalling the opportunity to win or avoid losing money were found to reinforce quicker and less variable RTs. These results suggest that the level of monetary incentive used in the current study was sufficiently motivating and engaged effortful response processes towards improved performance; although monetary gain and the avoidance of loss appeared equally reinforcing.

Our findings relating to the neural processing of reinforcement cues were mixed. We predicted that reinforcement-related cues would promote enhanced anticipatory brain activity as participants, motivated by monetary reinforcement, engaged attentional control and effortful processes in preparation to respond. Consistent with our prediction, cue-P3 was enhanced to cues of monetary gain, affirming the sensitivity of this component to the anticipation of monetary reward (Goldstein et al., 2006; Goldstein et al., 2008; Yeung & Sanfey, 2004), and highlighting the role of P3 in the allocation of attention (Polich & Kok, 1995) and motivational processes (Carrillo-de-la-Peña & Cadaveira, 2000; Groom et al., 2010). Indeed, consistent with a motivational interpretation, this component has been found to be attenuated in individuals who exhibit motivational deficits such as chronic cocaine users (Goldstein et al., 2008). Contrary to predictions,

cue P3 was not enhanced following cues relating to monetary loss. Nevertheless, evidence from fMRI work suggests that the anticipation of monetary reward may be especially salient, with increased activation in the nucleus accumbens during the anticipation of reward but not punishment (Knutson et al., 2001). Indeed support for a differential effect of monetary loss on anticipatory brain activity is mixed (Knutson et al., 2001; Stark, et al., 2011), and much of this research has focused on comparisons between gain and no-gain rather than loss per se (e.g. Bjork et al., 2004; Bjork et al., 2010; Bjork et al., 2008). Furthermore, with regards to the ERP literature, more work has explored the neural correlates of anticipatory brain activity associated with monetary reward of different magnitudes rather than loss (e.g., Goldstein et al., 2006; Goldstein et al., 2008). While there is some evidence of a greater effect of monetary loss relative to gain on cue P3 (Löw et al., 2008), other related work has not reported a consistent effect of loss on anticipatory brain activity (for a review see Brunia et al., 2011).

The predicted effects in relation to the CNV following cues of monetary gain or loss were not observed. In general, less work has assessed the effect of reinforcement on the CNV despite its clear role in effortful response preparation. Evidence from previous studies for an effect of monetary incentive on the CNV is mixed; for example, although one study found CNV to be modulated by reward (e.g. Pierson, et al., 1987), other work has not found this component to be influenced by either the opportunity to win or lose money (e.g. Goldstein et al., 2006). The disparity between these findings might be due to paradigm differences; Goldstein focused exclusively on variations in the level of monetary reward using a blocked task design whilst Pierson et al. (1987) employed a conditioning paradigm in which participants learnt the association between three tones and a monetary gain, loss and control condition. Goldstein et al. (2006) have also argued that personality traits might influence individual sensitivity to reward. Certainly Pierson et al. (1987) found a larger CNV differences in hedonic than anhendonic participants suggesting the capacity to experience pleasure may influence the sensitivity of the CNV to reward. In contrast to our predictions, but consistent with Goldstein et al. (2006), we did not find a differential effect of cues of potential monetary gain or loss on the CNV. It is possible that additional task design factors may have influenced these results, however here monetary gain and loss conditions were randomly presented whilst Goldstein et al. (2006) employed a blocked design. Future research should employ varying magnitudes of monetary reward and loss to examine these effects further.

Second, with regard to the neural processing associated with the target we predicted that, motivated by cues signalling the opportunity to win or avoid losing money, participants would engage additional

attentional resources to ensure effective processing of the target stimulus under these conditions. Unlike cue P3, the target P3 component was enhanced by *both* monetary gain and loss conditions relative to the feedback only control condition. This result serves as further evidence for the motivating effects of reinforcement on attention, and suggests that the effortful processes that are engaged during the anticipation of reinforcement are translated into enhanced attentional control to task-relevant stimuli as indexed by target P3. Although a differential effect of positive or negative reinforcement on target P3 was not identified in the current study, this finding warrants further investigation with larger samples and multiple levels of monetary incentive.

Third, in terms of neural processing associated with reinforcement outcome and more specifically feedback relating to the success or failure of task performance, our prediction that the FRN would be enhanced to negative feedback in the monetary loss relative to the feedback-only control condition was confirmed. This result is consistent with the notion that the FRN reflects a reinforcement learning signal, generated by the mesencephalic dopamine system, which acts as an alerting mechanism to indicate the occurrence of negative (or unexpected) salient outcomes such as actual monetary loss (Holroyd et al., 2006; Wu & Zhou, 2009; Yeung et al., 2005; Zhou, et al., 2010). Source localisation analyses suggest that the FRN is generated in the anterior cingulate cortex (ACC, Gehring & Willoughby, 2002), and this signal is thought to instigate subsequent monitoring strategies via the ACC to improve performance and subsequent outcomes (Holroyd & Coles, 2002). We also examined the LPP to successful and failed outcomes in the *monetary loss* and *gain* conditions, and predicted that the LPP would be enhanced to feedback indicating monetary gain (positive feedback, gain condition) and loss (negative feedback, loss condition) relative to the *feedback-only* control condition. The results support an enhanced LPP to monetary reward, consistent with other work showing enhanced positivity following win feedback (Wu & Zhou, 2009; Zhou et al., 2010). In the monetary loss condition, the LPP was enhanced following successfully avoided monetary loss, and there was a tendency for this component to be enhanced following actual monetary loss. The LPP has been consistently demonstrated to be enhanced by emotionally salient events such as pleasant and unpleasant IAPS pictures, emotional faces and reward (Hajcak et al., 2010). Yeung and Sanfey (2004) argue that this component is sensitive to high-level motivational evaluations such as disappointment and indeed, larger LPP amplitudes for negative compared with positive affective picture processing have often been reported (Weinberg & Hajcak, 2010). In contrast however, the findings pertaining to monetary reward and loss are less clear, with reports of a larger LPP response to both positive (Kamarajan, et al., 2009;

Yeung et al., 2005; Zhou et al., 2010) and negative reinforcement outcomes (De Pascalis et al., 2010). Much of this work however has incorporated unpredictable feedback in gambling-style tasks (e.g. Hajcak, et al., 2005) or tasks in which loss occurred more infrequently (De Pascalis et al., 2010). In the main analysis of the current study however, we examined successful and unsuccessful trials separately and therefore compared conditions of equal frequency. Here we found motivationally salient outcomes such as omitted loss and loss itself elicited larger LPP amplitudes, consistent with the notion that this component reflects higher order affective processing such as disappointment and relief. A direct comparison between the LPP following comparable gain and loss outcomes did not reveal any significant differences, suggesting an equivalent neural response relating to the affective processing of monetary gain and loss. Nevertheless future research should verify the sensitivity of the LPP to reinforcement valence with a range of reinforcement values and types (e.g. social vs. monetary reward).

The findings from the current study complement, and should be considered alongside other paradigms which have also examined electrophysiological correlates of reinforcement processing within the context of gambling (e.g., Marco-Pallares et al., 2008), observing another person's win or loss (Marco-Pallarés et al., 2010), and learning and adaptive behaviour (Cavanagh et al., 2010; Cohen & Ranganath, 2007). ERP and fMRI research using related paradigms have manipulated context to examine the behavioural relevance of reward-predicting cues (Nahum et al., 2011), decision-making following learned reward-predicting cue associations (Cohen et al., 2009), and attempted to dissociate reward probability and reward uncertainty (Yu et al., 2011). Other research has focused on electrophysiological methods as an effective tool to examine psychopathology-related alterations in sensitivity to reinforcement, including patients with depression (Bress et al., 2012; Foti & Hajcak, 2009), autism (Larson et al., 2011), ADHD (Holroyd et al., 2008), problem gamblers (Oberg et al., 2011) and adolescents who exhibiting risky behaviours (Crowley et al., 2009). Despite the large body of fMRI research utilising the MID task to dissociate the neural response to reinforcement anticipation and outcome, to the best of the authors' knowledge, only two other electrophysiological studies have utilised the eMID task to examine electrocortical responses to reinforcement (Cohen et al., 2012; Kawasaki et al., in press). These studies differ from the current study however in a number of important ways. First, both Cohen et al., (2012) and Kawasaki and Yamaguchi (in press) have focused on the synchronisation of EEG frequency bands (theta and beta) during the task and not the event-related brain response to reinforcement cues or feedback. Second, Kawasaki and Yamaguchi (in press) modified the eMID task to include a visual working memory

task instead of the simple target detection task and examined theta and beta amplitudes during the retention interval under different reward-schedules finding an association between both theta and beta amplitude and visual working memory capacity. Third, in a very interesting application of the MID task, Cohen et al. (2012) examined electrophysiological data recorded directly from the NAcc and from the cortex (via scalp EEG) in patients undergoing deep brain stimulation surgery for the treatment of obsessive compulsive disorder (OCD). These authors focused on reward anticipation and found that top-down directed synchrony between the NAcc and EEG activity over the medial frontal cortex (MFC; i.e. MFC → NAcc) was increased during the anticipation of a reward relative to a no reward condition, while there was little effect of reward condition on bottom-up synchrony (i.e. NAcc → MFC; Cohen et al., 2012). These data suggest the MFC might modulate reinforcement processing in the NAcc, however the generalisability of these findings to healthy participants needs to be considered in future research. The current study then, builds on these and previous studies using alternative paradigms, by examining reinforcement-related modulation of the ERP response during reward anticipation *and* outcome. Further, by using the original and elegantly simple eMID task design, the currents study avoids many of the confounds associated with the engagement of multiple task-related processes (such as attention, inhibition and visual working memory, see Kawasaki & Yamguchi, in press) and individual differences related to task difficultly.

Nevertheless, this is the first study to use the e-MID task to decompose the event-related neural response to the anticipation and outcome of reinforcement, as well as reinforcement-contingent target processing and future research should explore the current findings further and extend the current task design. First, consistent with more traditional versions of the MID task used in fMRI paradigms, future ERP research should modify the current task to include a range of incentive magnitudes to examine the parametric effect of reward/loss on neural responses to monetary reinforcement. Second, to explore the relationship between reinforcement and decision making, this work could be extended to a battery of tasks examining the effect of real and hypothetical reward, alternative types of reward such as social reinforcement, reward predictability and expectancy, the influence of temporal factors such as a delay prior to reward receipt, and the influence of agency. Third, the ERP work in the current study could also be complemented by simultaneously recorded fMRI to localise these neural responses to specific brain generators. Fourth, although robust performance and ERP differences were not observed for two samples, these effects should be explored further with a larger sample, broader age-range and gender matched samples. Fifth, while IQ scores were available for the adolescent group, no IQ measure was taken for the

young adult group, although they were all undergraduate University students. The association between IQ and reinforcement sensitivity should be considered further. Sixth, there were minor differences in the version of the e-MID task performance by the two samples. Specifically, the inter-stimulus interval between the cue and target was fixed for the adult sample (2000 ms) and jittered for the adolescent sample (varying randomly between 2000 and 2500 ms). Although we found no differences in the CNV of the adult and adolescent sample, it may be possible that a variable inter-stimulus interval (jittering makes the onset of the target less predictable) may have had an effect on the CNV. Future research should replicate these findings. Finally, this work should be extended to clinical groups characterised by motivation deficits, such as ADHD and substance use disorders.

In summary, the current paper demonstrates the utility of the e-MID task as an effective method with which to decompose the neural chronology of positive and negative reinforcement processing during anticipatory cue, target and outcome stages. Although monetary gain and loss conditions shared some common elements in terms of associated brain potentials (e.g. target P3, LPP) the findings highlight the special motivational salience of monetary gain at anticipatory (cue P3), target (target-P3) and outcome stages (LPP) of processing monetary reinforcement. More particularly, these data support the use of the e-MID task as a complement to standard fMRI paradigms in the dissection of normal and abnormal neural responses to reinforcement.

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Table and Figure captions

Table 1. Mean (SD) SNR for each ERP component in Feedback only, Monetary gain and Monetary Loss conditions

Table 2. Statistics and mean (SD) task performance and ERP data to the cue, target and feedback stimuli.

Figure 1. Schematic of task timing and ERP component and task performance data examined

Figure 2. ERP data to the cue and target stimuli. Cue P3 in the centroparietal region, CNV in the frontocentral region and Target P3 in the centroparietal region for the Feedback-only control condition (solid line), Monetary Gain (dotted line) and Monetary Loss (dashed line) conditions. ERP waveforms have been averaged across age group. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis. Topographic maps across condition are also shown.

Figure 3. ERP data to feedback stimuli. The FRN at frontocentral sites in the negative feedback condition (left), the LPP at centroparietal sites in the negative (middle) and positive (right) feedback conditions. ERP waveforms have been averaged across age group. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis. Topographic maps across condition are also shown.

Supplementary Figures – Appendix A to F

Figure 4. Appendix A. Adolescent (left) and young adult (right) Cue P3 data at central, central/parietal, parietal and occipital midline electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis

Figure 5. Appendix B. Adolescent (top) and young adult (below) CNV data at left hemisphere, midline and right hemisphere sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis

Figure 6. Appendix C. Adolescent (left) and young adult (right) Target P3 data at central, central/parietal, parietal and occipital midline electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis

Figure 7. Appendix D. Adolescent (left) and young adult (right) FRN data following negative feedback at frontal and frontocentral electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds **along the x-axis**

Figure 8. Appendix E. Adolescent (left) and young adult (right) LPP data following positive feedback at central, central/parietal, parietal and occipital midline electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis

Figure 8. Appendix E. Adolescent (left) and young adult (right) LPP data following negative feedback at central, central/parietal, parietal and occipital midline electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis

Table 1.

	Control SNR M(SD)	Gain SNR M(SD)	Loss SNR M (SD)
Cue P3	3.58 (1.99)	3.60 (1.96)	3.25 (1.26)
CNV	3.03 (1.14)	3.13 (1.50)	2.86 (1.07)
Target P3	4.56 (2.31)	4.71 (1.88)	4.61 (1.87)
FRN Neg FB	3.22 (1.70)	3.53 (1.58)	3.77 (1.64)
LPP Pos FB	3.74 (2.03)	4.70 (1.93)	4.39 (2.17)
LPP Neg FB	3.62 (1.69)	4.04 (1.71)	4.47 (1.83)

Note: Neg FB, Negative feedback; Pos FB, Positive feedback

Table 2.

	Comparison	***Details***	***F***	***df***	***p***
Monetary Gain					
MRT (ms)	G vs. C	203 (27) < 215 (19)	18.92	1,37	.001
SD of RT (ms)	G vs. C	48 (25) < 68 (32)	19.05	1,37	.001
Cue P3 (μV)	G vs. C	8.91 (5.63) > 6.61 (5.24)	13.69	1,31	.001
CNV (μV)	G vs. C	-1.13 (4.68) > -0.56 (3.60)	1.32	1,31	ns
Target P3 (μV)	G vs. C	13.21 (6.50) > 11.76 (5.03)	4.26	1,32	.047
FRN (μV)	Neg FB: No G vs. C	6.77 (6.22) > 6.82 (5.92)	<1	1,31	ns
LPP (μV)	Pos FB: G vs. C	13.36 (8.03) > 8.10 (5.59)	24.90	1,33	.001
LPP (μV)	Neg FB: G vs. C	16.62 (7.14) > 14.84 (6.40)	2.18	1,31	ns
Monetary Loss					
MRT (ms)	L vs. C	205 (22) < 215 (19)	11.65	1,37	.002
SD of RT (ms)	L vs. C	55 (32) < 68 (32)	4.99	1,37	.032
Cue P3 (μV)	L vs. C	7.38 (5.33) > 6.61 (5.24)	1.31	1,31	ns
CNV (μV)	L vs. C	-1.28 (3.63) > -0.56 (3.60)	2.63	1,31	ns
Target P3 (μV)	L vs. C	14.04 (6.13) > 11.76 (5.03)	14.77	1,32	.001
FRN (μV)	Neg FB: No G vs. C	4.51 (6.57) > 6.82 (5.92)	5.09	1,31	.031
LPP (μV)	Pos FB: L vs. C	11.71 (6.53) > 8.10 (5.59)	22.63	1,33	.001
LPP (μV)	Neg FB: L vs. C	16.92 (7.07) > 14.84 (6.40)	3.74	1,31	.062

Note: G, Gain; C, Control; Neg FB, Negative feedback; Pos FB, Positive feedback

TIME: →

Event:	**Cue**	**ISI**	**Target**	**ISI**	**Positive Feedback**	**Negative Feedback**
	Feedback only control: blue cross Monetary Gain: blue octagon Monetary Loss: blue diamond		White square		Green '✓'	Red '✗'
Task timing:	250 ms	2000 - 2500 ms*	250 ms	1450 ms	1500 ms	1500 ms
ERP component:	Cue P3		Target P3			FRN
	CNV				LPP	LPP
Comparison:	G or L vs. C		G or L vs. C		Gain vs. omitted gain	Loss vs. avoided loss

Note: * ISI between cue and target was fixed (2000 ms) for the young adult group. ISI, inter-stimulus interval; CNV, contingent negative variation; LPP, Late positive potential; FRN, feedback-related negativity; G, Gain; L, Loss; C, Feedback only control.

Figure 1.

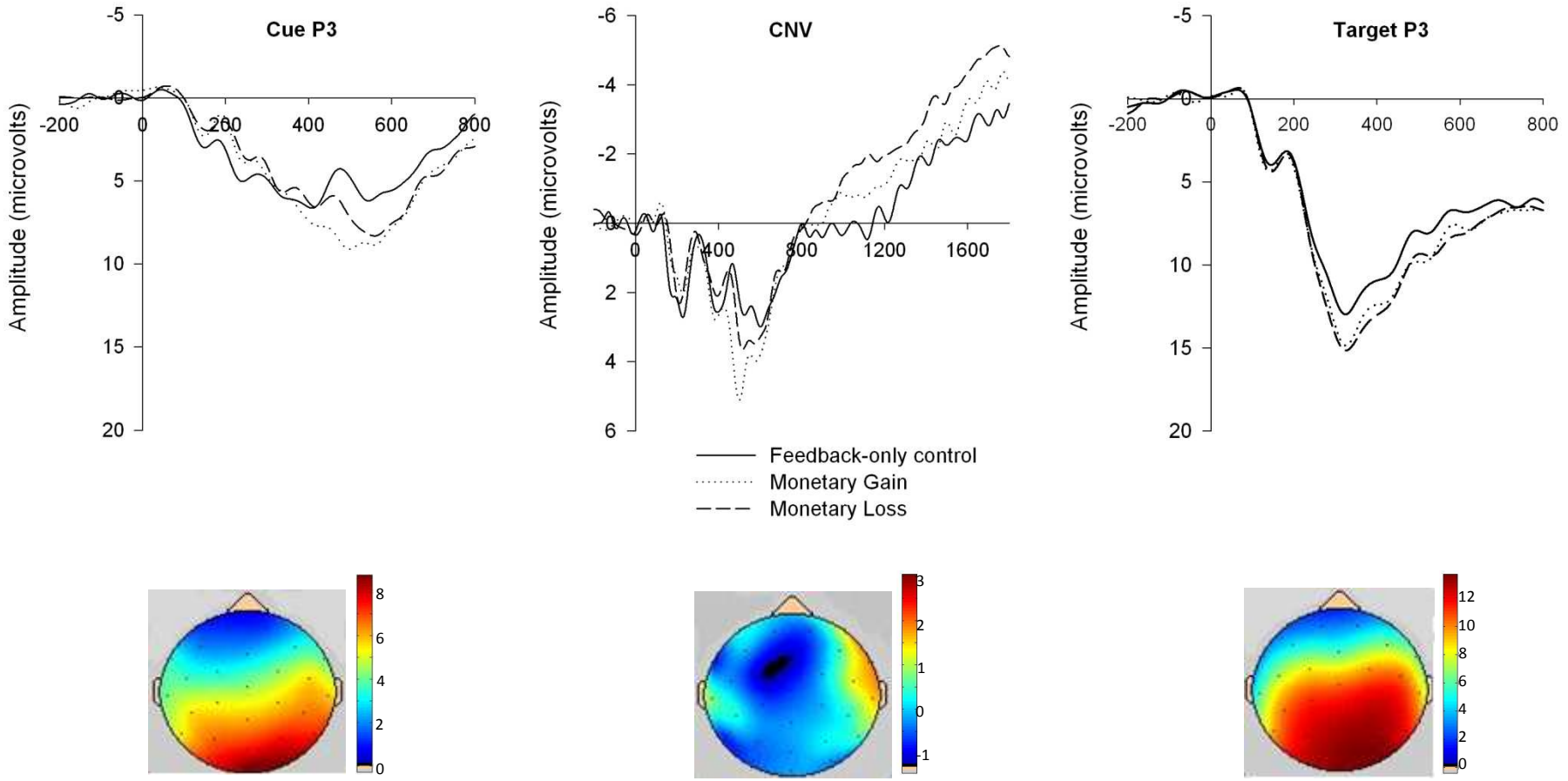
Cue P3
CNV
Target P3
Amplitude (microvolts)
-200
0
200
400
600
800
1200
1600
Feedback-only control
Monetary Gain
Monetary Loss

Figure 2.

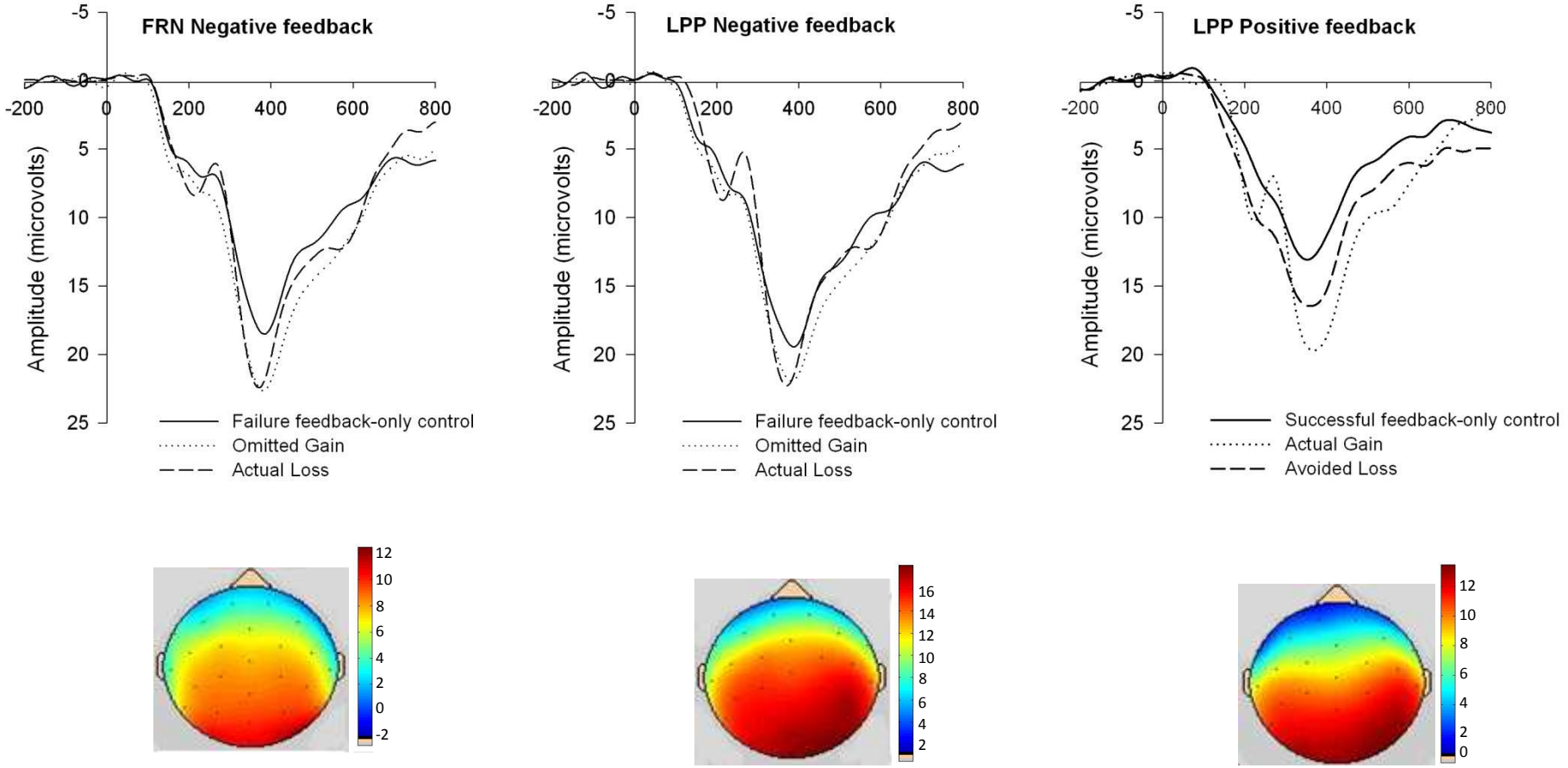
FRN Negative feedback
LPP Negative feedback
LPP Positive feedback
Amplitude (microvolts)
-5
0
5
10
15
20
25
-200
0
200
400
600
800
Failure feedback-only control
Omitted Gain
Actual Loss
Successful feedback-only control
Actual Gain
Avoided Loss
12
10
8
6
4
2
0
-2
16
14
12
10
8
6
4
2

Figure 3.

Appendix A: Supplementary ERP figures – Cue P3

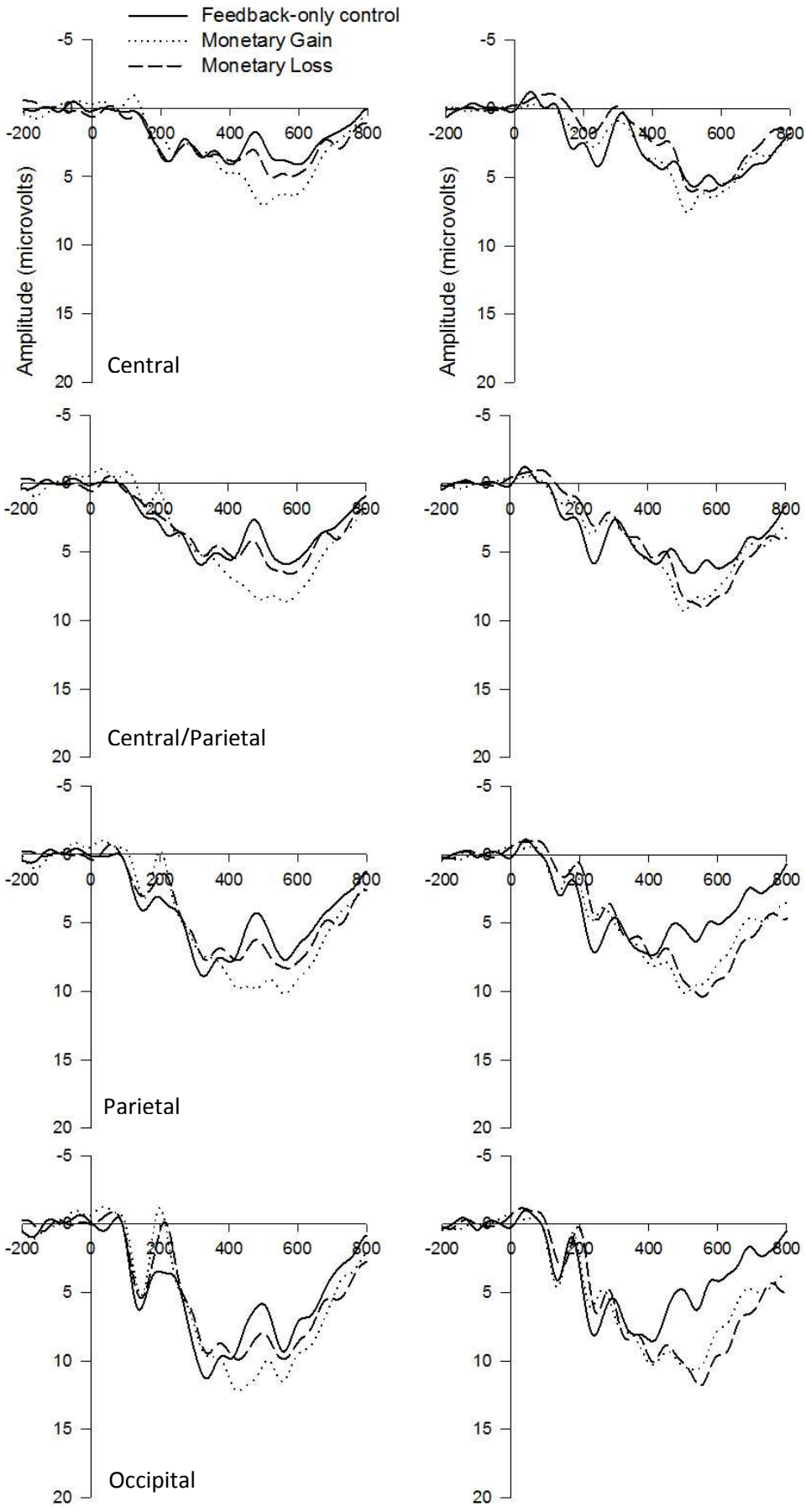

Figure 4. Appendix A. Adolescent (left) and young adult (right) Cue P3 data at central, central/parietal, parietal and occipital midline electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis.

Appendix B: Supplementary ERP figures – CNV

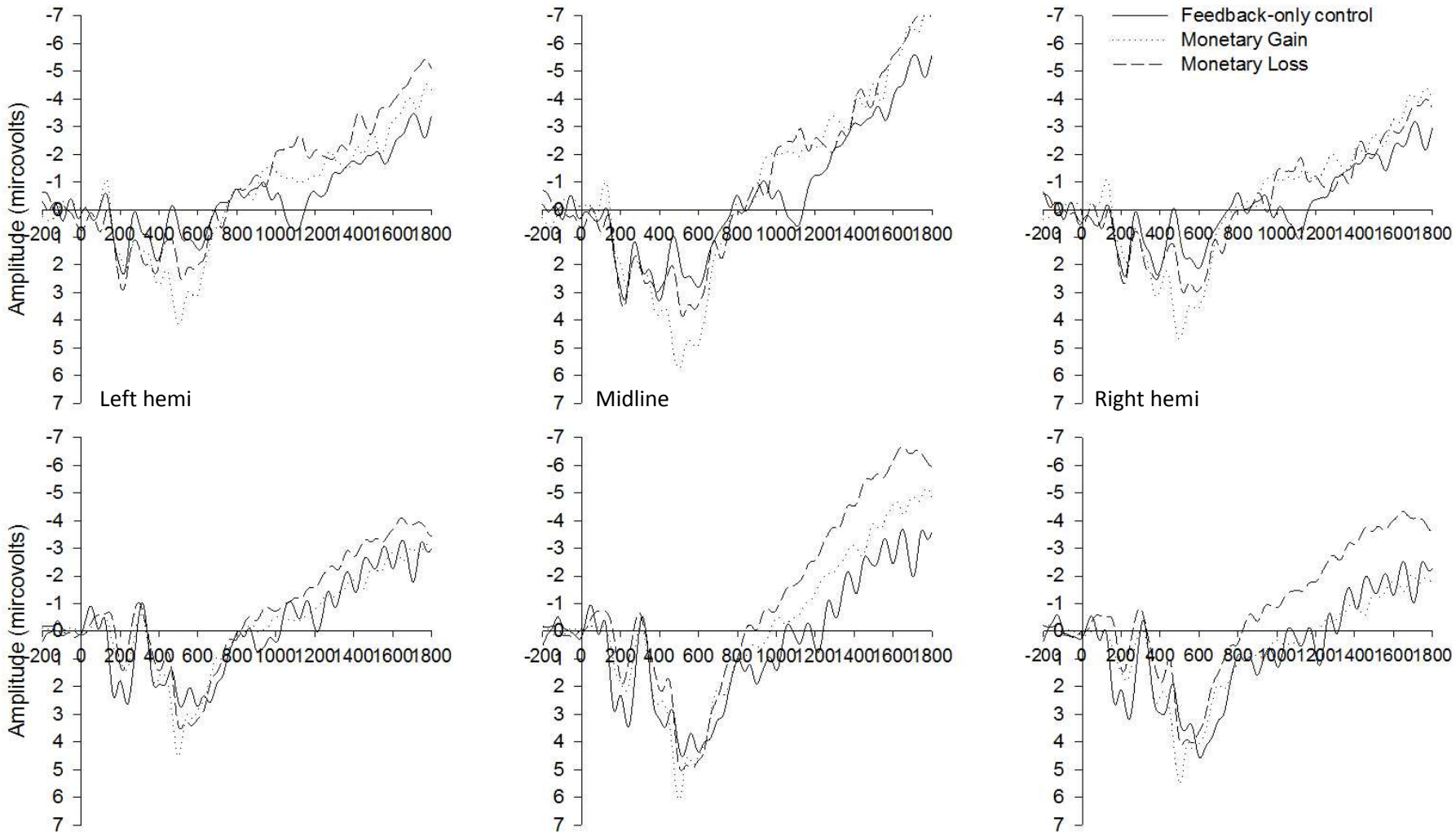

Figure 5. Appendix B. Adolescent (top) and young adult (below) CNV data at left hemisphere, midline and right hemisphere sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis

Appendix C: Supplementary ERP figures – Target P3

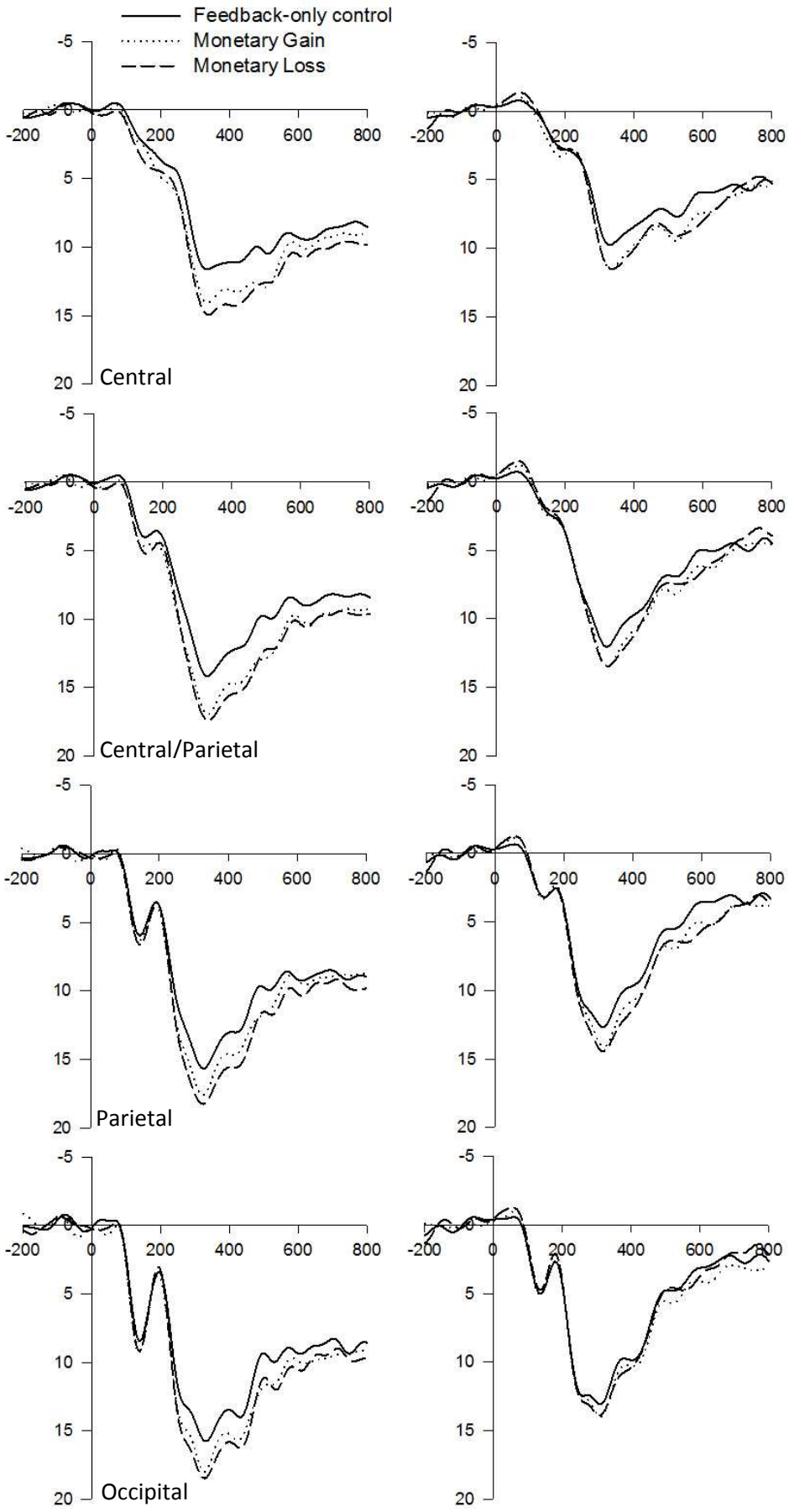

Figure 6. Appendix C. Adolescent (left) and young adult (right) Target P3 data at central, central/parietal, parietal and occipital midline electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis

Appendix D: Supplementary ERP figures – FRN following negative feedback

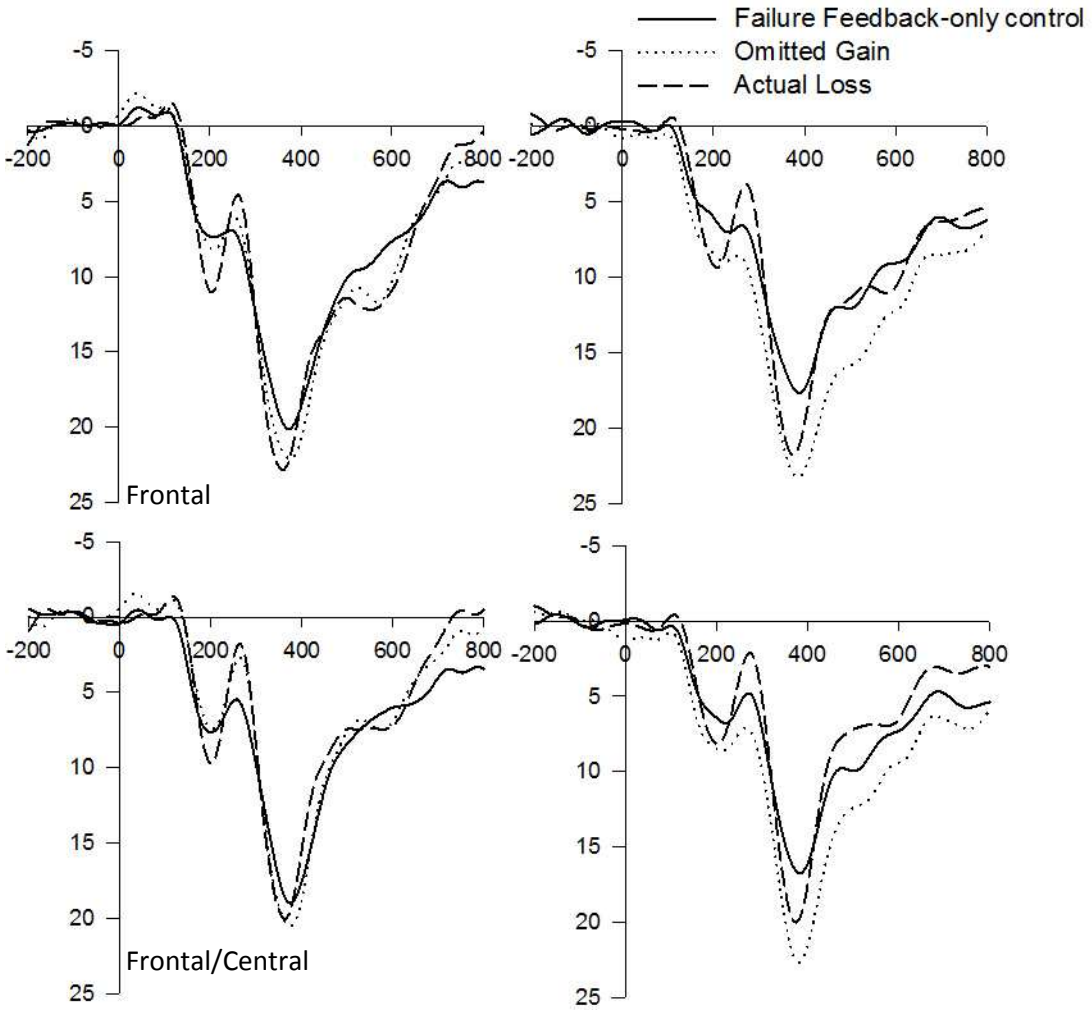

Figure 7. Appendix D. Adolescent (left) and young adult (right) FRN data following negative feedback at frontal and frontocentral electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis

Appendix E: Supplementary ERP figures – LPP following positive feedback

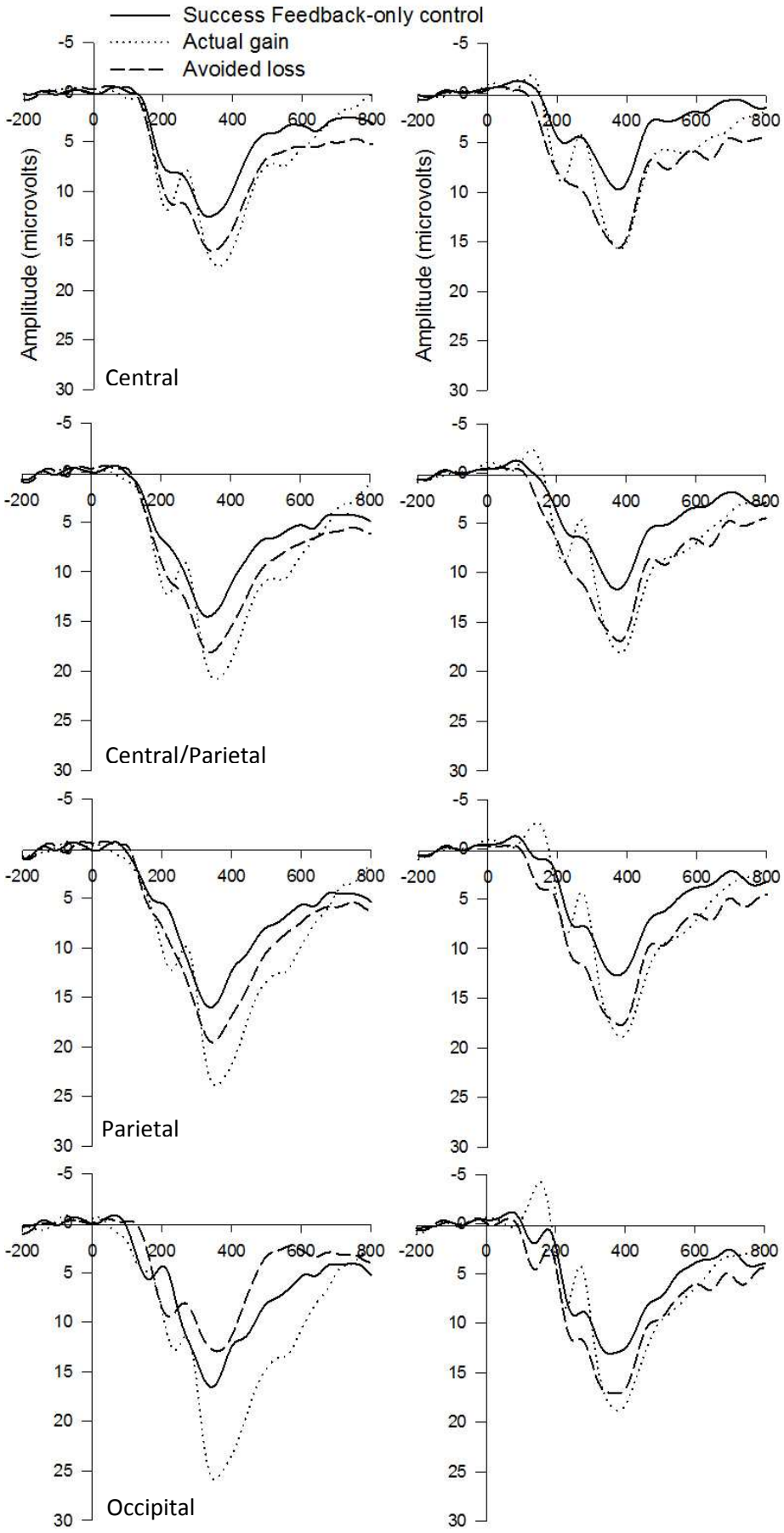

Figure 8. Appendix E. Adolescent (left) and young adult (right) LPP data following positive feedback at central, central/parietal, parietal and occipital midline electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis

Appendix F: Supplementary ERP figures – LPP following negative feedback

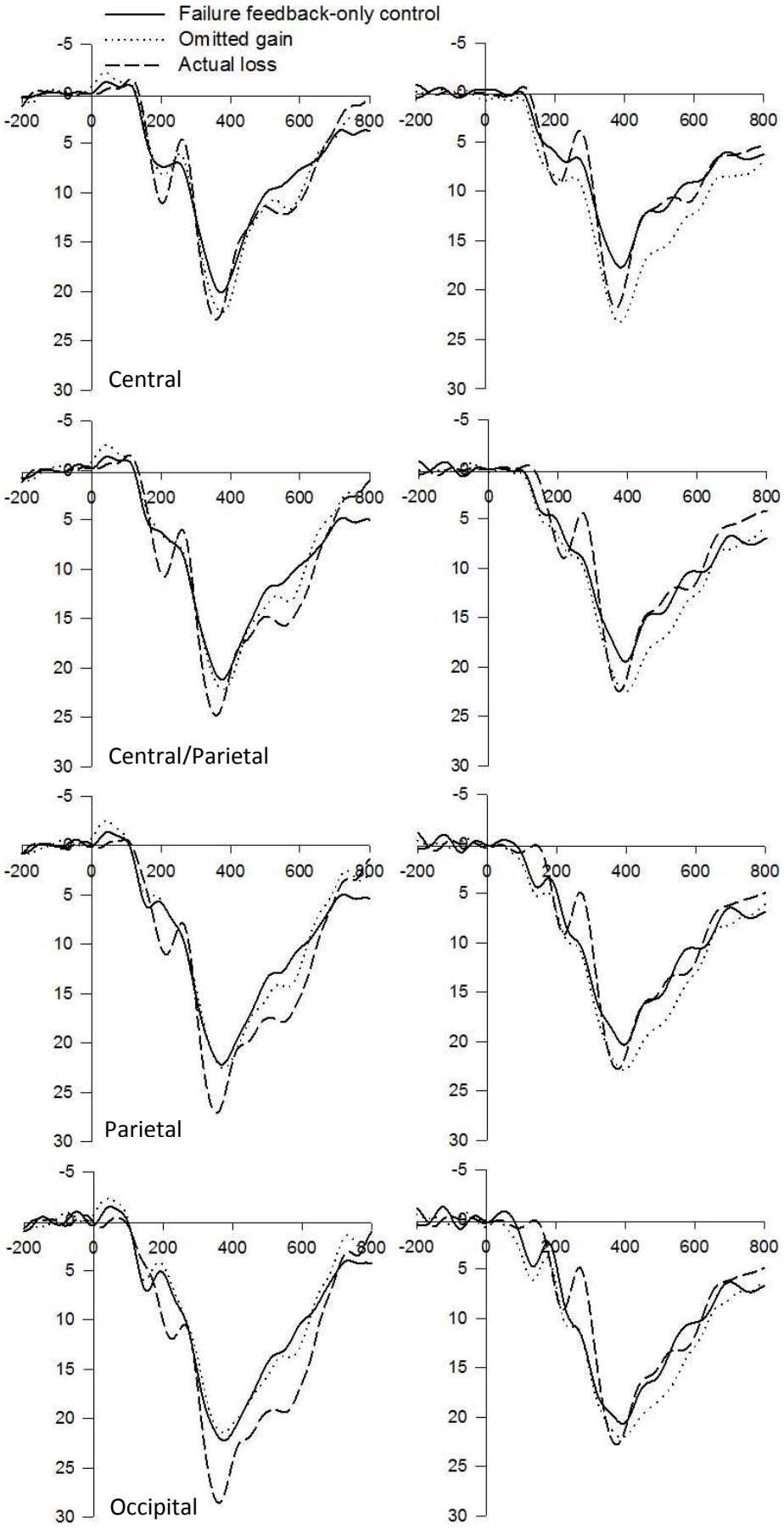

Figure 9. Appendix F. Adolescent (left) and young adult (right) LPP data following negative feedback at central, central/parietal, parietal and occipital midline electrode sites. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis